

# NAVAL POSTGRADUATE SCHOOL

## Monterey, California



# THESIS

## MODELING MAN-MADE EPIDEMICS

by

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**MODELING OF MAN-MADE EPIDEMICS**

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Submitted in partial fulfillment of the  
requirements for the degree of

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# ABSTRACT

This thesis develops a mathematical model to explore epidemic spread through the Ground Combat Element (GCE) of the Marine Expeditionary Unit (MEU). The model will simulate an epidemic caused by a biological attack using an agent that has the ability to spread through person-to-person contact (small pox, hemorrhagic fever, etc.) A stochastic modeling process will be used along with widely accepted mathematical formulas for an SEIR (Susceptible-Exposed-Infectious-Removed) epidemic model. A heterogeneous population composed of numerous homogenous subgroups with varying interaction rates simulates the unique structure of military combat units. The model will be evaluated to determine which units facilitate the most rapid spread of the epidemic. The model will then test a number of different scenarios to determine the effects of varying quarantine techniques, vaccination strategies and protective postures on the spread of the disease.

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## **DISCLAIMER**

The computer program in the Appendix is supplied on an “as is” basis, with no warranties of any kind. The author bears no responsibility for any consequences of using this program.



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# I. INTRODUCTION

It is better to have an approximate answer to the right question than a right answer to the wrong question. John Tukey [Ref. 1: pp. 4-5]

## A. RESEARCH OBJECTIVES

This thesis will describe and model the spread of a man-made epidemic within a closed population that contains arbitrarily many subgroups. The primary population to be modeled is the Marine Expeditionary Unit (Special Operations Capable)(MEU(SOC)). Military combat units have a population and mixing structure that is very different from the general population. The spread of an epidemic intentionally introduced into a military population will differ greatly from the naturally occurring epidemics in the general public. This thesis will use a susceptible, exposed, infectious, removed (SEIR) epidemic model to estimate the overall progression of an epidemic introduced into a military combat unit, specifically the ground combat element (GCE) of the MEU(SOC).

A series of factors determine the effects of a biological weapon delivered upon any population. Factors that determine the effect of a biological weapon include, but are not limited to, the type and amount of agent, dissemination methods, atmospheric conditions that effect virulence and infectivity, dilution in the atmosphere and the protective posture of the exposed population. [Ref. 2: pp. 30-32] The Soviet Union spent many years studying the effect these factors have on the success of their weapons. They developed a measure called the specific expenditure value, or  $Q_{50}$ .  $Q_{50}$  is the amount of the agent that needs to be delivered, to infect no less than fifty percent of the target population, that is evenly distributed over 1 km<sup>2</sup>. This measure took into account all of the above factors and was obviously situationally dependent.[Ref. 3: p. 21]

The purpose of this research is not to incorporate all of the variables involved with developing a biological weapons effect model. The necessary knowledge

of physics, molecular biology, meteorology and many other disciplines is beyond the scope of this thesis. This research is specifically interested in developing a model to estimate the effect of a biological weapon that possesses the ability to produce secondary infections through the transmission of a communicable disease. The model will then begin from the point after an effective  $Q_{50}$  event has occurred. We are interested in the spread of the epidemic throughout the entire population after one small subset has been infected at the fifty percent level.

After the model is formulated numerous test cases will be run to develop a baseline database that will contain runs where the epidemic is allowed to spread unhindered. This data will be used to validate that the model is actually working as expected. The model will allow the user to insert specific defensive techniques and advance the epidemic at any rate they choose. The effect of these defensive techniques will then be compared to the initial data to determine if there is any reduction in the overall spread of the epidemic. As epidemiology is a study of many variables the output of the model is not expected to produce mathematically exact answers. The purpose is to show that such a tool may be useful to operating forces in planning their response to biological weapons.

## **B. BACKGROUND**

### **1. The Study of Epidemics**

The study of epidemics is as old as the study of medicine. Hippocrates, in the fifth century B.C., suggested that there were many things that may cause disease in humans and by keeping track of the circumstances surrounding each case of disease, a doctor may be able to infer some causal effects. Keeping track of rates of disease in the different seasons, different communities, among different ages and sexes and different lifestyles may give clues to what is causing a certain disease.

It was John Grant, in 1662, who did the first quantitative study of disease patterns in a population. Through weekly study of birth and death reports in London

he found that males had both a higher birth and mortality rate. He was also able to track the seasonal change in mortality rates that Hippocrates had mentioned more the 2000 years prior. [Ref. 4: pp. 4-6]

Some of the earliest work in a developing deterministic system to address the spread of an epidemic came from W.O. Kermack and A.G. McKendrick. They saw every population (of fixed size =  $N$ ) at any time divided into three stages or cohorts with respect to the epidemic:[Ref. 5: pp 312-313]

$S(t)$  = susceptible

$I(t)$  = infected and circulating throughout the population

$R(t)$  = removed by recovery, quarantine or death

$$S(t) + I(t) + R(t) = N = \text{constant} \quad (\text{I.1})$$

At  $t = 0$ ,

$$S(0) + I(0) = N \quad (\text{I.2})$$

## 2. The Use of Disease as a Weapon

Biological weapons do not have an extensive history of use in the world of military tactics. While the effects can be devastating, biological agents still require time to incubate, spread and reach an incapacitating state. Attacking the enemy with plague in the middle of a fierce conflict will do little in the immediate future to affect the outcome of the battle. One of the first recorded uses of disease as an offensive weapon came from the fourteenth century when the Tafta army, in present day Crimea, catapulted the bodies of plague victims over the walls of Kaffa during a siege. The Japanese attempted to use plague against the Chinese before and during World War II. They released billions of plague infected fleas over their target areas in an attempt to conceal the attack as a natural occurrence of plague. The effectiveness of the attack was never quantified as the official Japanese position is that they never used such a weapon. [Ref. 3: p. 166] In 1984 the Rajneesh religious sect in the United States used the food borne parasite Salmonella in an attack. Their attack was an

effort to disrupt the upcoming local elections. Using food service areas in restaurants, offices and public venues they managed to infect over 700 people. Reportedly there were no fatalities. [Ref. 6] A covert attack on the staging and port facilities used during Operations Desert Shield and Desert Storm could have had a significantly different outcome. In the world of instant news, reports of a plague or small pox outbreak would have significantly hindered the public relations fight and crippled the logistical build up of troops.

Most recently, weapons grade anthrax has been mailed to several liberal politicians and media personalities. To date a total of 18 people have been infected with either pulmonary or cutaneous forms of anthrax. Five cases became fatal. Shortly after the second confirmed case of anthrax surfaced, it became apparent that there had been a deliberate release of anthrax. Many medical professionals began hypothesizing about the repercussions of the release of a communicable disease rather than the non-communicable anthrax.

[I]f obtained and intentionally released, smallpox could cause a public health catastrophe because of its communicability. Even a single case could lead to 10 to 20 others. It is estimated that no more than 20% of the population has any immunity from the prior vaccination. There is no acceptable treatment, and the communicability by aerosol requires negative-pressure isolation. Therefore, these limited isolation resources in medical facilities would be easily overwhelmed.[Ref. 7: pp. 1-7]

Since the events surrounding the terrorist attacks on 11 September 2001, biological weapons have once again been thrust to the center of national attention. The threat is not just from terrorism. As the United States military takes action in Afghanistan, evidence has been discovered that al Queda and the Taliban are researching the development of chemical and biological weapons. Our service men and women are again on the front lines with the threat of biological weapons looming over them.

Of the three types of weapons of mass destruction; Nuclear, Biological and Chemical; the Biological Weapons family is the easiest to produce. The industrial

infrastructure, scientific know-how and available technology are readily available to any country with a moderately sophisticated pharmaceutical industry. The tools and techniques for developing a biological agent are identical to those used in developing many of the vaccines widely used all over the world today. Manufacturing the agent and turning it into a military weapon is another process all together. Many agents are fragile and susceptible to environmental stresses such as heat, cold, humidity, ultra-violet light and many others. There are processes and techniques to enhance the survivability of biological agents. The former Soviet Union developed ways to keep even the most fragile organisms alive during the delivery process.

Biological weapons can most assuredly be placed on strategic missiles, cruise missiles and combat aircraft. These assets are not, however, necessary for the deployment of such weapons. Crop dusters, car bombs, delivery trucks and even suitcase bombs can be used to deliver significant amounts of a biological agent. [Ref. 8: pp. 39-40]

### **3. The Soviet Biological Weapons Program**

The Convention on the Prohibition of the Development, Production and Stockpiling of Bacteriological (Biological) and Toxin Weapons and on Their Destruction (Biological Weapons Convention(BWC)) signed in 1972, prohibited the development of biological agents for use in offensive weapons. The Soviet Union, a signer of the treaty, almost immediately stood up Biopreparat(one of four agencies inside the Soviet Union that produced biological weapons). Biopreparat was responsible for developing, testing and weaponizing various agents for use as biological weapons against the United States. In his book *Biohazard*, Dr Ken Alibek, former Deputy Director of Biopreparat and defector from the Soviet Union, spells out the build up of the Soviet biological weapons program.

Over a twenty-year period that began, ironically, with Moscow's endorsement of the Biological Weapons Convention in 1972, the Soviet Union built the largest and most advanced biological warfare establishment in the

world. We were among the 140 signatories of the convention, pledging "not to develop, produce, stockpile of otherwise acquire or retain" biological agents for offensive military purposes. At the same time, through our covert program, we stockpiled hundreds of tons of anthrax and dozens of tons of plague and smallpox near Moscow and other Russian cities for use against the United States and its Western allies. [Ref. 3: p. x]

The Soviet Union, as late as 1992, was also developing and weaponizing genetically engineered smallpox plague, Marburg, and Venezuelan equine encephalitis (VEE). The specific expenditure values ( $Q_{50}$ ) for these weapons is very small.

Specific Expenditure Value ( $Q_{50}$ )	
Agent	Amount
Smallpox	3.0 – 3.5kg/sq.km
Plague	3.5 – 4.0kg/sq.km
Marburg	0.2 – 0.8kg/sq.km
VEE	3.0 – 3.5kg/sq.km

[Ref. 9]

Since its inception, the Soviet biological weapons program searched for unique agents that would be difficult to identify and more difficult to treat.

The Soviet government decided that the best agents were those for which there was no known cure. This shaped the entire course of our program and thrust us into a never-ending race against the medical profession. Every time a new treatment or vaccine came to light somewhere, we were back in our labs, trying to figure out how to overcome its effects.[Ref. 3: p. 18]

In 1989 the Soviet Union had completed work on a new type of agent. They had found a way to genetically engineer the pneumonic strain of plague to include a myelin toxin in the bacteria's DNA that attacked the central nervous system. The toxin attacks the coating of nerve receptors, the myelin sheath, causing paralysis. In one agent, the Soviet Union now had the ability to release the world's oldest biological weapon with a decidedly new and dangerous twist.

Although the toxin-plague was never developed into a weapon the technology of combining bacteria with naturally occurring toxins set the stage for an entire new class of weapons, the genetically engineered biological weapon. [Ref. 3: p. 167]

#### **4. The Marine Expeditionary Unit (Special Operations Capable)**

The Marine Expeditionary Unit (Special Operations Capable) (MEU(SOC)) is the standard forward-deployed Marine expeditionary organization (see Figure 1). Their mission is to be a forward presence able to respond within days to crises in their area of operation. There are always at least three MEUs(SOC) deployed at any given time. One in the Mediterranean, one in the western Pacific and one in the Indian Ocean or Arabian Gulf region. Each MEU consists of a standing command element(CE), a ground combat element (GCE), an aviation combat element (ACE) and a combat service support element(CSSE). This model is going to be concerned specifically with the GCE.

The GCE is comprised of a reinforced infantry battalion or battalion landing team. Standard reinforcements include an artillery, reconnaissance, engineer, armor, assault amphibian units, and other detachments as required. The MEU CE retains some flexibility in deciding the number and type of attachments the GCE will require. [Ref. 10: p. 2.4]

This model is based on Battalion Landing Team 1/8 which deployed to the Mediterranean from Nov 1996 through May 1997.

### **C. ORGANIZATION**

This thesis contains five chapters, including this introduction. Chapter II describes the mathematics of epidemiology and introduces accepted formulae for both deterministic and stochastic epidemic modeling. Chapter III explains the layout of the SEIR model used for this research. User defined parameters, the Microsoft Excel spreadsheets, operation of the program and the mathematics behind the program are



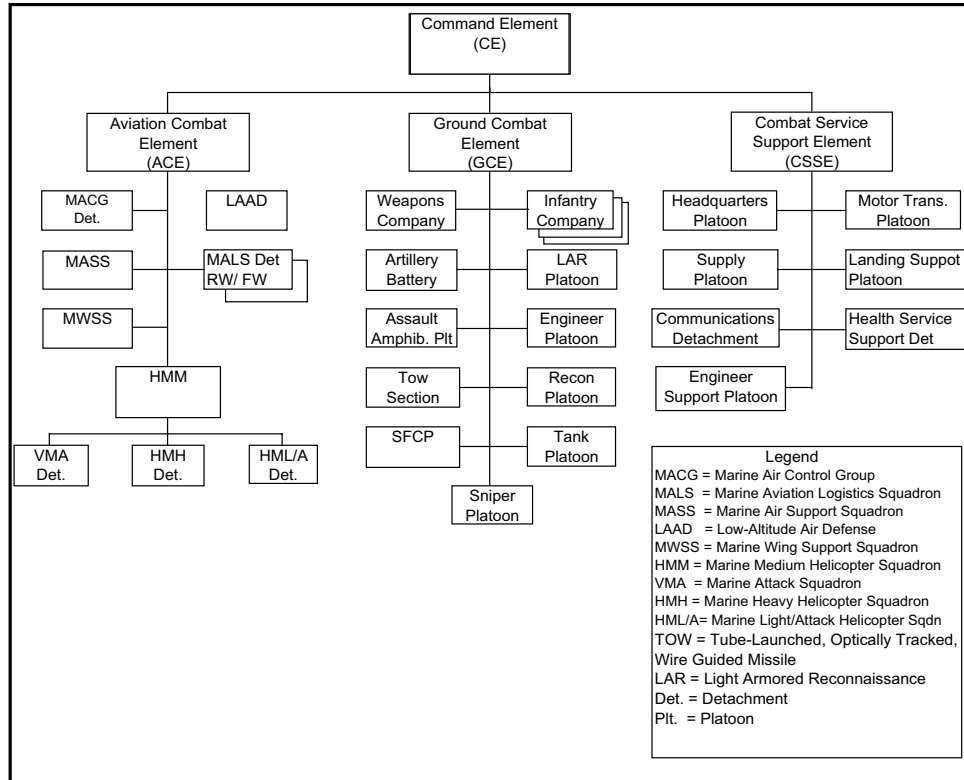


Figure 1. Marine Expeditionary Unit (Special Operations Capable)

all explained here. Chapter IV introduces the scenario that was tested, produces the results of the unhindered epidemic spread trials, explains different defensive techniques and their effect on the overall spread of the epidemic. Chapter V presents a summary of the research and recommendations for areas of further research.

## II. THE MATHEMATICS OF EPIDEMICS

The first idea that must occur to anyone who hears me is the place which among all such subjects we shall gratefully assign to mathematical science. George Buchanan at the Epidemiological Society of London, 1881 [Ref. 1: pp. 4-5]

Basic epidemic models allow for variations in the different stages of the infection. Identifying the stages to include depends on the dynamics of the disease, the composition of the population and the length of the model the researcher is developing. An individual can be in any one of the stages of infection. Susceptible (S), the individual is able to contract the infection; exposed (E), the individual has contracted the disease but is not yet infectious or symptomatic; infectious (I), the individual is contagious and may or may not be showing symptoms; and removed (R), an individual can be removed from the population by recovering with immunity, being quarantined or by death. In addition to those above some models include stage M, a passive immunity stage reached only by the birth of an infant who temporarily holds immunity through vertical transmission of the mother's antibodies. The name of the model is an acronym of the above letters describing the flow patterns between cohorts. Some of the most commonly studied models are MSEIR, MSEIRS, SEIR, SEIRS, SIR, SIRS, SEI, SEIS, SI, and. [Ref. 11: p. 601]

### A. THE BASIC EQUATIONS

Kermack's and McKendrick's work on the closed population (a population of finite size, with no new entries) epidemic problem has been the springboard for much study in the field of epidemiology. In a closed population it was assumed that individuals became infected at a rate proportional to the number of susceptible and the number of infected, individuals became removed at a rate proportional to the number of infected. In a closed population it was not possible to enter the susceptible or leave the removed categories. This lead to the famous Kermack-McKendrick (K

and K) equations.

$$\begin{aligned}
\dot{S} &= -\beta SI \\
\dot{I} &= \beta SI - \gamma I \\
\dot{R} &= -\gamma I
\end{aligned}
\tag{II.1}$$

A stochastic process forms naturally from the deterministic K and K equations above. The process will be Markovian with a finite number of states (at most  $N + 1$ ). There are four possible transitions involved in the process during a small time interval  $(t, t + dt)$ :

1. an infection ( $S \rightarrow S - 1$  and  $I \rightarrow I + 1$ ) with probability  $\beta SI dt + o(dt)$ ;
2. a removal ( $I \rightarrow I - 1$  and  $R \rightarrow R + 1$ ) with probability  $\gamma I dt + o(dt)$ ;
3. a variety of multiple transitions, with total probability  $o(dt)$ ; and
4. no change, with probability  $1 - (\beta S + \gamma) I dt + o(dt)$ . [Ref. 12: p. 154]

In both the deterministic and stochastic systems  $\beta$  is the infection rate and  $\gamma$  is the removal rate. The ratio,  $\rho = \frac{\gamma}{\beta}$ , is called the epidemic threshold or relative removal rate. The size of the epidemic is therefore simply a function the size of the population and the size of the threshold ratio.[Ref. 13: p. 178]

The previous two systems make a number of assumptions about the population. They are:

1. The population is closed, there are never any new susceptible individuals entering the population.
2. The population is homogeneous, there is an even mixing of all members and every member is equally likely to contract the disease.
3. Every meeting of an infected host with a susceptible individual leads to a new infection.
4. There are only three possible classifications of members; susceptible, infective and removed. [Ref. 12: p. 156]

These assumptions make the systems rather easy to work with, precise solutions can be found to both the deterministic and stochastic systems. The assumptions however do not model precisely, the interactions of most populations or the behavior of most diseases. Most populations are arranged in various subgroups and clusters of subgroups which have varying interaction rates and susceptibilities to infection. Individuals may also belong to more than one subgroup or mixing group at the same time. The infective stage of the disease may also be broken down into a number of smaller stages that will have an effect on the contact rate an individual has with others. Most diseases have a latent period in which the individual is infected with the disease but not yet contagious and not yet symptomatic. The disease will also have an incubation period which is measured from the time of infection until the surfacing of the first symptoms. The most dangerous time of the infective period is the difference in time from the end of the latent period to the end of the incubation period. During this time the individual can transmit the disease to a susceptible individual but has no symptoms to warn him that he is a danger to others. This time period is well known to all who study the AIDS epidemic. The average latent period is just about 11 months while the incubation period can be up to twelve times as long. Studies in the late 1980's and early 1990's identified the mean incubation period of AIDS in gay males to be  $11.70 \pm .40$  years.[Ref. 1] [Ref. 14: pp. 21-33] This dramatic difference between the end of the latent period and the end of the incubation period have made controlling the AIDS epidemic a formidable task. A military weapon that captured this large difference in latent and incubation periods, for example a latent period of 3 days and an incubation period 30 days, would be very effective. As the individual moves further into the symptomatic portion of the infection his effective contact rate will drop dramatically. There is, indeed, much more variability in the spread of an epidemic than the K and K equations will allow.

## B. THE HETEROGENEITY OF EPIDEMICS

Most populations are not a simple well mixed group of homogeneous individuals. A population may be divided up into a series of overlapping subgroups and clusters of subgroups. The amount of interaction within the group varies from subgroup to subgroup. The amount of interaction between subgroups again depends on the type of groups one is considering.

In their 1974 article "Stochastic Simulation Models for Two Immunization Problems", Elveback, Fox and Ackerman develop an influenza model based on the structure of a small suburban community. They divided their population up into families (with and without children), age groups (pre-school, grade school, high school, young adult, older adult), play groups and family clusters. The subgroups were obviously not distinct as an eight-year-old second-grader would belong to a family with children, the grade school subgroup, some play group and a family cluster.

For their model they developed a number of variabilities in the parameters of the epidemic which were meant to be general enough to adapt for all infectious agents that spread through person-to-person contact. These will be adopted and added to for this research. The variations include:

1. Variations in relative susceptibility between individuals or subgroups
2. Variations in the length of the latent period.
3. Variations in the length of the incubation period.
4. Variations in the length of the infectivity period.
5. Ability to assign what proportion of infections actually become symptomatic.
6. Variations in the time of withdrawal from the susceptible population following infection, as a function of subgroup assignment.

They introduce a parameter to describe the contact rate,  $\beta$ , between any two individuals in the population. This is the rate at which any two individuals make contact sufficient enough to pass the infection. During each iteration of time the

program considers each susceptible individual separately, where his probability of remaining infection free depends on numerous parameters.

$$P(t)(\text{probability person } i \text{ escapes infection on day } t) = e^{-S_i R_i} \text{ where}$$

$$R_i = \sum^G [\beta_{ig} \sum^{I_g} \theta_c]$$

$$G = \text{the number of the individual's mixing group}$$

$$I_g = \text{the number of infective cases in group } g$$

$$\theta_c = \text{the relative infectiouness of case } c$$

$$\beta_{ig} = \text{the contact rate for person } i \text{ in group } g$$

$$S_i = \text{the relative susceptibility of person } i$$

[Ref. 15: pp. 92-95]

## C. CURRENT STUDIES

Currently a number of government agencies including the new Office of Homeland Defense are interested in estimating the effects of Biological Warfare and Bioterrorism. John Bombardt of the Institute for Defense Analyses (IDA) authored, "Contagious Disease Dynamics for Biological Warfare and Bioterrorism Casualty Assessment." He studied a 1995 Ebola Hemmoragic Fever (EHF) outbreak in the Democratic Republic of the Congo. Armed with the data from the EHF outbreak, Bombardt attempts to estimate, as a function of time, the health care and mortuary services needed to deal with a military attack or terrorist incident using an agent similar to EHF.

Bombardt uses the Haydon-Woolhouse-Kitching (HWK) SEIR Algorithm, which was initially used to model the dynamics of foot and mouth disease in cattle herds in the United Kingdom. The HWM SEIR Algorithm introduces a set of four finite difference equations.

$$S[n] = S[n-1] - P[n-1]\delta t, \tag{II.2}$$

$$E[n] = E[n-1] + (P[n-1] - P[n - \alpha_a - 1])\delta t, \quad (\text{II.3})$$

$$I[n] = I[n-1] + (P[n - \alpha_a - 1] - P[n - \zeta_a - 1])\delta t, \quad (\text{II.4})$$

$$R[n] = R[n-i] + P[n - \zeta_a - 1]\delta t; \text{ and} \quad (\text{II.5})$$

$$P[n] = \xi[n]S[n]I[n] \quad (\text{II.6})$$

$$N_0 = S[n] + E[n] + I[n] + R[n] \quad (\text{II.7})$$

In equations *II.3 – II.5*  $\alpha_a$  represents the sojourn time in the exposed stage of the disease and  $\zeta_a$  represents the sojourn time in the exposed and infectious stages. The sojourn time is the amount of time an individual spends in a specific stage of the disease. The function  $\xi[n]$  is a time varying disease transmission rate.

The function  $P[n]$  describes the interaction between the cohort groups. This function inserts a non-linearity into the system of equations so that changes in the initial conditions cannot easily be used to predict the outcome of the epidemic. For each implementation of the algorithm the researcher would have to determine the function  $P[n]$  from some piece of data. For the EHF outbreak Bombardt used recorded dates of symptomatic onset and Monte Carlo trials to derive an expression for the average new infections per unit time. [Ref. 16: pp. 2-7]

Combining the above models and epidemic theory we will now develop a model to describe the spread of a man made epidemic through the GCE of a Marine Expeditionary Unit.

### III. THE SEIR MODEL

The scenario used in the development of this model is that the MEU is ashore conducting operations when one or two members of a platoon present to their battalion aid station with symptoms of some disease. The medical professionals then make the assumption that the member has been exposed to a biological agent. The agent is diagnosed to be one that is communicable. The commanders now must decide what steps to take to limit the spread of the disease and to maintain the mission capability of their unit.

#### A. POPULATION DYNAMICS OF COMBAT UNITS

The population dynamic of military combat units lead to a unique type of mixing within the population. The hierarchical structure of the military means that units do the majority of their mixing with those units directly above or below themselves in the command structure. There are service and support elements that mix with all units but the majority of mixing happens along chain of command lines. It is this unique population dynamic that this model attempts to imitate.

An assumption about the size of a homogeneous unit has to be established before implementing the model. A homogeneous unit is one where the unit is considered well-mixed, there is an equal likelihood that contact will be made between any pair of members. The size of this unit depends directly on the overall size of the population. Military units have a self-similar force structure. Negating headquarters units, attachments and reinforcements each military unit is comprised of 3 to 4 subordinant units whose structure is similar to the parent unit. For example the Marine infantry regiment contains three infantry battalions, which contain four infantry companies, which each contain three infantry platoons, which each contain three infantry squads, which contain three infantry fire-teams, which each contain three infantry men (plus a fire-team leader). For this model we are dealing with the reinforced infantry bat-



talion, the largest homogeneous unit is assumed to be the platoon. Using this scaling the regimental model would assume a company to be a homogeneous unit.

## B. WHY EXCEL?

The choice to use Microsoft's Excel program was made because of its wide availability and use inside the military. Ideally, the program was designed so that someone familiar with the interactions of the combat unit and a medical professional could together define the necessary parameters for the model. They can then estimate when an attack occurred, which unit/units were affected and then progress the epidemic to study the possible extent of the spread of the disease. Parameters can be changed at any time to simulate different protective steps.

## C. THE PARAMETERS

The program opens with two initial worksheets, "Population Characteristics" and "Agent Characteristics" (see Figure 2) where the user fills in the necessary initial information on the population and suspected biological agent. The following parameters are necessary to run the program:

Unit:	Name of unit
Size:	Number of members of the unit
ECR:	Effective Contact rate, entered as a percentage (0,1)
Virulence:	Entered as a percentage (0,1)
Latent Period:	The mean latent period entered in integer form.
Incubation Period:	The mean incubation period entered in integer form.

The effective contact rate will be the mean percentage of the homogeneous unit that any one member has sufficient contact with to pass along the disease. Virulence is defined as the ability of a virus to cause an infection. For the purpose of this model it will be used to describe the mean percentage of effective contacts that will lead to a new exposure. The latent period of a disease is the time in which the disease is

	A	B	C	D	E	F	G								
1	<b>Company</b>	<b>Platoon</b>	<b>Size</b>	<b>ECR</b>											
2	H&S Company	BNCP	124	0.75		<div style="border: 1px solid black; padding: 5px; width: fit-content;">           Create Interaction Matrix and Epidemic Progression Table         </div>									
3		HCOM	30	0.5											
4		HSER	63	0.5											
5		HMED	67	0.5											
6	Company A	AHQ	14	0.75											
7		A1	50	0.75											
8		A2	50	0.75											
9		A3	51	0.75											
10		AW	25	0.5											
11	Company B	BHQ	14	0.75		<div style="border: 1px solid black; padding: 10px;"> <b>Agent Characteristics</b> <table border="1" style="width: 100%; border-collapse: collapse;"> <tr> <td style="width: 70%;">Name of Agent:</td> <td>Test</td> </tr> <tr> <td>Virulence:</td> <td style="text-align: center;">0.75</td> </tr> <tr> <td>Latent Period:</td> <td style="text-align: center;">5</td> </tr> <tr> <td>Incubation Period:</td> <td style="text-align: center;">10</td> </tr> </table> </div>	Name of Agent:	Test	Virulence:	0.75	Latent Period:	5	Incubation Period:	10	
Name of Agent:	Test														
Virulence:	0.75														
Latent Period:	5														
Incubation Period:	10														
12		B1	50	0.75											
13		B2	50	0.75											
14		B3	51	0.75											
15		BW	25	0.5											
16	Company C	CHQ	14	0.75											
17		C1	50	0.75											
18		C2	50	0.75											
19		C3	51	0.75											
20		CW	25	0.5											
21	Weapons Company	WHQ	6	0.75											
22		WMOR	69	0.75											
23		WAA	50	0.75											
24		WHMG	28	0.75											
25	Attachments	ATY	139	0.75											
26		CEP	36	0.75											
27		TNK	20	0.75											
28		SSP	30	0.75											
29		RCN	30	0.75											
30		LAR	45	0.75											
31		AAV	55	0.75											
32															

Figure 2. Population and Agent Characteristics Worksheets

dormant, the individual is not contagious. The incubation period is the time prior to which symptoms appear.

”Contact rate” can be changed at anytime during the execution of the model. All other parameters remain fixed until the process is reset. An assumption has been made that the onset of diagnosable symptoms occurs in the day following the end of the incubation period. An individual will therefore be removed from the active population the day symptoms appear.

## D. THE INTERACTION MATRIX

Upon entering the necessary parameters the user then clicks the ”Create Interaction Matrix and Epidemic Progression Table” button and a series of new sheets are

	BNCP	HCOM	HSER	HMED	AHQ	A1	A2	A3	AW	BHQ	B1	B2	B3	BW	CHQ	C1	C2	C3	CW	WHQ	WMOR	WAA	WHMG	ATY	CEP	TNK	SSP	RCN	LAR	AAV
BNCP																														
HCOM	10																													
HSER	10	10																												
HMED	10	10	10																											
AHQ	9	2	5	2																										
A1					7																									
A2					7	3																								
A3					7	3	3																							
AW					7	3	3	3																						
BHQ	9	2	5	2	3																									
B1						7																								
B2						7	3																							
B3						7	3	3																						
BW						7	3	3	3																					
CHQ	9	2	5	2	3					3																				
C1											7																			
C2											7	3																		
C3											7	3	3																	
CW											7	3	3	3																
WHQ	9	2	5	2	5					5					5															
WMOR																7														
WAA																7	3													
WHMG																7	3	3												
ATY	7	2	5	2	2					2					2					2										
CEP	5	2	5	2	2					2					2					2										
TNK	5	2	5	2	2					2					2					2										
SSP	5	2	5	2	2					2					2					2										
RCN	5	2	5	2	2					2					2					2										
LAR	5	2	5	2	2					2					2					2										
AAV	5	2	5	2	2					2					2					2										

Figure 3. Population and Agent Characteristics Worksheets

produced. One is called "Interaction Matrix." This is a lower triangular matrix with the platoon names entered on the initial worksheet as the row and column headings. (see Figure 3) The user must then enter the expected number of contacts between units to occur in a 24 hour period. This must be an integer entry. If the units are not expected to have at least one contact per day the user may leave blank or enter zero. The entries in the interaction matrix can be changed at any time during the process to simulate controls placed by higher headquarters or periods of varying interactivity rates.

## E. EPIDEMIC PROGRESSION

The Epidemic Progression sheet is the main working sheet for this model. The sheet has four main areas; unit totals (See Figure 4), action buttons, population totals (See Figure 5) and a hidden area. The unit totals area keeps the daily count of

Unit	Size	S(t)	E(t)	I(t)	R(t)	% Available
BNCP	124	62	37	16	9	0.927419355
HCOM	30	24	6	0	0	1
HSER	63	58	5	0	0	1
HMED	67	62	5	0	0	1
AHQ	14	12	2	0	0	1
A1	50	50	0	0	0	1
A2	50	50	0	0	0	1
A3	51	51	0	0	0	1
AW	25	25	0	0	0	1
BHQ	14	11	3	0	0	1
B1	50	50	0	0	0	1
B2	50	50	0	0	0	1
B3	51	51	0	0	0	1
BW	25	25	0	0	0	1
CHQ	14	9	4	1	0	1
C1	50	50	0	0	0	1
C2	50	50	0	0	0	1
C3	51	51	0	0	0	1
CW	25	25	0	0	0	1
WHQ	6	3	3	0	0	1
WMOR	69	69	0	0	0	1
WAA	50	50	0	0	0	1
WHMG	28	28	0	0	0	1
ATY	139	134	5	0	0	1
CEP	36	33	2	1	0	1
TNK	20	19	1	0	0	1
SSP	30	30	0	0	0	1
RCN	30	27	3	0	0	1
LAR	45	39	6	0	0	1
AAV	55	53	2	0	0	1

Figure 4. Epidemic Progression: Unit Totals

how many members of each unit are currently in each of the different disease stages (Susceptible, Exposed, Infectious and Removed). There are three different action buttons the user can employ from this page. "Advance Time" cycles the programs algorithms and updates daily totals as if 24 hours had passed. "Reset this page" resets the current page back to day zero and removes all data. This will allow the user to do multiple runs with the same initial conditions to compare quantitative results. "Reset Entire Program" returns the user back to the initial worksheet. This needs to be done if the user wants to change any of the disease parameters or change the composition of his population. At any time while operating the Epidemic Progression sheet the user can change any of the values on the Interaction Matrix or change any of the effective

<b>Day</b>		<b>9</b>	
<b>Advance Time</b>		<b>Population Totals</b>	
<b>S(t)</b>	<b>E(t)</b>	<b>I(t)</b>	<b>R(t)</b>
1251	84	18	9
<b>Reset this page</b>			
<b>Reset Entire Program</b>			
<b>Name of Agent:</b>	Test		
<b>Virulence:</b>	0.75		
<b>Latent Period:</b>	5		
<b>Incubation Period:</b>	10		

Figure 5. Epidemic Progression: Population Totals and Action Buttons

contact rates on the Population Characteristics sheet. The population totals area has a simple day counter and contains the population totals for the different stages of the disease. The hidden area holds the daily progression totals for the exposed and infectious stages of the disease. Let  $L$  be the entry in the latent period and  $C$  be the difference between the entry in the incubation period and the entry in the latent period. The hidden area will contain  $L + C + 2$  columns,  $L + 1$  of those columns will be exposure columns and  $C + 1$  will be infectious columns.

## F. THE ALGORITHMS

The algorithms that drive this model are broken up into three major sections. The homogeneous epidemic spread, heterogeneous epidemic spread and total epidemic

progression.

## 1. Homogeneous Epidemic Spread

Homogeneous epidemic spread is used to describe the disease dynamics inside a homogeneous unit. Individuals in the unit move between the four different disease classes  $S \rightarrow E \rightarrow I \rightarrow R$  through a combination of deterministic K and K equations and a Markov process. The following variables are introduced to describe this progression.

$$\begin{aligned}
v &= \text{virulence} \\
r &= \text{effective contact rate} \\
L &= \text{latent period} \\
C &= \text{incubation period - latent period} \\
N &= \text{size of unit} \\
S(t) &= \text{number of susceptible at time } t \\
E(t) &= \text{total number of exposed at time } t \\
I(t) &= \text{total number of infectious at time } t \\
R(t) &= \text{number of removed at time } t
\end{aligned} \tag{III.1}$$

The exposed stage,  $E(t)$ , and infectious stage,  $I(t)$ , is individuals that have been exposed or infectious for varying numbers of days.

$$E(t) = E_1(t) + E_2(t) + \cdots + E_{L+1} \tag{III.2}$$

$$I(t) = I_1(t) + I_2(t) + \cdots + I_{C+1} \tag{III.3}$$

The subscripts indicate the number of days individuals have been in the exposed or infectious state.  $E_3(t)$  is the number of individuals that have been in the exposed state for 3 days. Movement between the days of the exposed and infectious

states is a Markov process with the following transition matrix.

	$E_1$	$E_2$	$\cdots$	$E_{L-2}$	$E_{L-1}$	$E_L$	$E_{L+1}$	$I_1$	$\cdots$
$E_1$	0	1	$\cdots$	0	0	0	0	0	$\cdots$
$E_2$	0	0	$\cdots$	0	0	0	0	0	$\cdots$
$\vdots$	$\vdots$	$\vdots$	$\vdots$	$\vdots$	$\vdots$	$\vdots$	$\vdots$	$\vdots$	$\vdots$
$E_{L-2}$	0	0	$\cdots$	0	1	0	0	0	$\cdots$
$E_{L-1}$	0	0	$\cdots$	0	0	0.8	0	0.2	$\cdots$
$E_L$	0	0	$\cdots$	0	0	0	0.4	0.6	$\cdots$
$E_{L+1}$	0	0	$\cdots$	0	0	0	0	1	$\cdots$
$I_1$	0	0	$\cdots$	0	0	0	0	0	$\cdots$
$\vdots$	$\vdots$	$\vdots$	$\vdots$	$\vdots$	$\vdots$	$\vdots$	$\vdots$	$\vdots$	$\vdots$

... continued

	$\cdots$	$I_1$	$I_2$	$\cdots$	$I_{C-2}$	$I_{C-1}$	$I_C$	$I_{C+1}$	$R$
$\vdots$	$\vdots$	$\vdots$	$\vdots$	$\vdots$	$\vdots$	$\vdots$	$\vdots$	$\vdots$	$\vdots$
$I_1$	$\cdots$	0	1	$\cdots$	0	0	0	0	0
$I_2$	$\cdots$	0	0	$\cdots$	0	0	0	0	0
$\vdots$	$\vdots$	$\vdots$	$\vdots$	$\vdots$	$\vdots$	$\vdots$	$\vdots$	$\vdots$	$\vdots$
$I_{C-2}$	$\cdots$	0	0	$\cdots$	0	1	0	0	0
$I_{C-1}$	$\cdots$	0	0	$\cdots$	0	0	0.8	0	0.2
$I_C$	$\cdots$	0	0	$\cdots$	0	0	0	0.4	0.6
$I_{C+1}$	$\cdots$	0	0	$\cdots$	0	0	0	0	1
$R$	$\cdots$	0	0	$\cdots$	0	0	0	0	1

An assumption for this model is that once an individual reaches the end of the incubation period and becomes symptomatic he is removed from the population by the beginning of the next day. There is no recovery where the individual can be reentered into the active population to full duty. Now the four equations for the progress of each state of the epidemic can be defined

$$S(t+1) = S(t) - \frac{vrS(t)I(t)}{N} \quad (\text{III.4})$$

$$E(t+1) = E(t) + \frac{vrS(t)I(t)}{N} - (.2(E_{L-1}(t)) + .6(E_L(t)) + E_{L+1}(t)) \quad (\text{III.5})$$

$$I(t+1) = I(t) + (.2(E_{L-1}(t)) + .6(E_L(t)) + E_{L+1}(t)) - (.2(I_{C-1}(t)) + .6(I_C(t)) + I_{C+1}(t)) \quad (\text{III.6})$$

$$R(t+1) = R(t) + (.2(I_{C-1}(t)) + .6(I_C(t)) + I_{C+1}(t)) \quad (\text{III.7})$$

## 2. Heterogeneous Epidemic Spread

The heterogeneous spread of the epidemic is based on how the different units interact with one another. The user will have established this through the interaction matrix by entering the mean number of expected effective contacts between each pair of units. These parameters can change at any time in the modeling process. An assumption for this model is that all members of the units are equally likely to take part in this interaction. This may not be exactly accurate as officers and staff noncommissioned officers are much more likely to attend staff meetings and planning sessions than a lower ranking enlisted Marine. There are, however, working parties and police details that are chosen at random and these interactions are significant.

The calculation of the amount of spread between units is then quite simple. For example, assume Unit i and Unit j are expected to have 10 interactions each day. One member is randomly selected from each unit and they are brought together for an interaction. The algorithm for determining the number of new exposures for both units is as follows:

New Exposures for Unit A = 0

New Exposures for Unit B = 0

For  $i = 1$  to 10

Randomly select on member from Unit A =  $A_i$

Randomly select on member from Unit B =  $B_i$

If  $A_i = S$  and  $B_i = I$  then



New Exposures for Unit A = New Exposures for Unit A +1

If  $A_i = I$  and  $B_i = S$  then

New Exposures for Unit B = New Exposures for Unit B +1

All other combinations lead to no change in new exposures.

Next  $i$

All possible unit combinations are checked and a hidden sheet tracks and totals all of the new exposures from 24 hours worth of interactions. These new exposures will be added to the new exposures from the homogeneous epidemic spread and begin the next day as  $Exp_{new}$ .

### **3. Totals**

Each time the "Advance Time" button is activated, the homogeneous spread and heterogeneous spread calculations occur using the previous days total. After all calculations are complete and the number of new exposures is totaled the unit totals are all advanced one day in preparation for the next advancement of time. The totals presented in the Epidemic Progression page are the totals for the end of the current day.

## IV. SCENARIO TESTING AND DATA

The scenario tested for this research is a typical mission for the GCE of the MEU(SOC). The Battalion Landing Team (BLT) has been inserted to secure an airfield and set-up a defensive posture to allow for the introduction of follow-on forces. A total of 1362 Marines and sailors are ashore operating in a defensive operations. Typical attachments and reinforcements have been made, local resistance is minimal but the situation is still very unstable. There has been intelligence that some biological weapons work has been ongoing in the country but no definite threat.

Although the BLT is self sufficient for up to 15 days there is often contact with the local population for interpreters, garbage removal and some transportation. At some time,  $t = 0$ , a biological agent is released, covertly, and one unit becomes exposed. The BLT then continues on with its daily activities until the epidemic becomes evident.

### A. UNHINDERED EPIDEMIC SPREAD

The first step in testing the model was to allow the epidemic to progress unhindered for 10 days. The only action taken against the epidemic was to remove the actively symptomatic individuals from the circulating population. Various initial conditions (see Table 1) were tested to develop a baseline database. The conditions tested included, initial unit exposed, level of that exposure, virulence, latent period and incubation period. Each possible combination of conditions was tested five times for a ten day period. Over 18,600 daily iterations were recorded in developing the database.

Upon completing the 18,600 iterations of the epidemic progression program, data analysis was performed on the entire set of data and selected scenarios. The mean ten day exposure level was 23.90% with a standard deviation of .131. This means that, throughout all different possible combinations of initial conditions, the

Various Initial Conditions Used in Testing Epidemic Model			
Virulence	Latent and Incubation Period	Unit Initially Exposed	Level of Initial Exposure
.25	2/4	Battalion Command Post (BNCP)	25%
	2/5		
.33	2/6	Rear Command Post (BNRR)	50%
	2/7		
.50	2/8		
	2/9		
.67	2/10	Alpha Company (ACO)	
	3/5		
.75	3/6	Charlie Company Reinforced (CCO+)	
	3/7		
	3/8		
	3/9		
	3/10	Weapons Company (WepCO)	
	5/10		

Table 1. Initial Conditions

average percentage of the total population that became exposed to the epidemic, after ten days of unhindered spread, was 23.90%. The distribution of these ten day exposure percentages can be seen in Fig 6.

After analyzing the entire data set, specific scenarios were extracted to allow examination of the progress of the epidemic when specific conditions were allowed to vary. The scenarios allowed the initial conditions to be held constant while one was allowed to vary.

## 1. Variations in Incubation Periods

For the first scenario the latent period was fixed at two days and the virulence was fixed at 67%. The initial unit exposed and exposure level were also fixed although two different units and exposure levels were investigated. For the battalion command post, exposed at 50%, and Alpha company exposed at 25%, we examined the incubation periods of 4, 5, 6, 7, 8, 9 and 10. The results are found in Table 2.

The trend is obvious at first glance, as the incubation period increases so does the percentage of exposure for the entire population (for latent/incubation periods

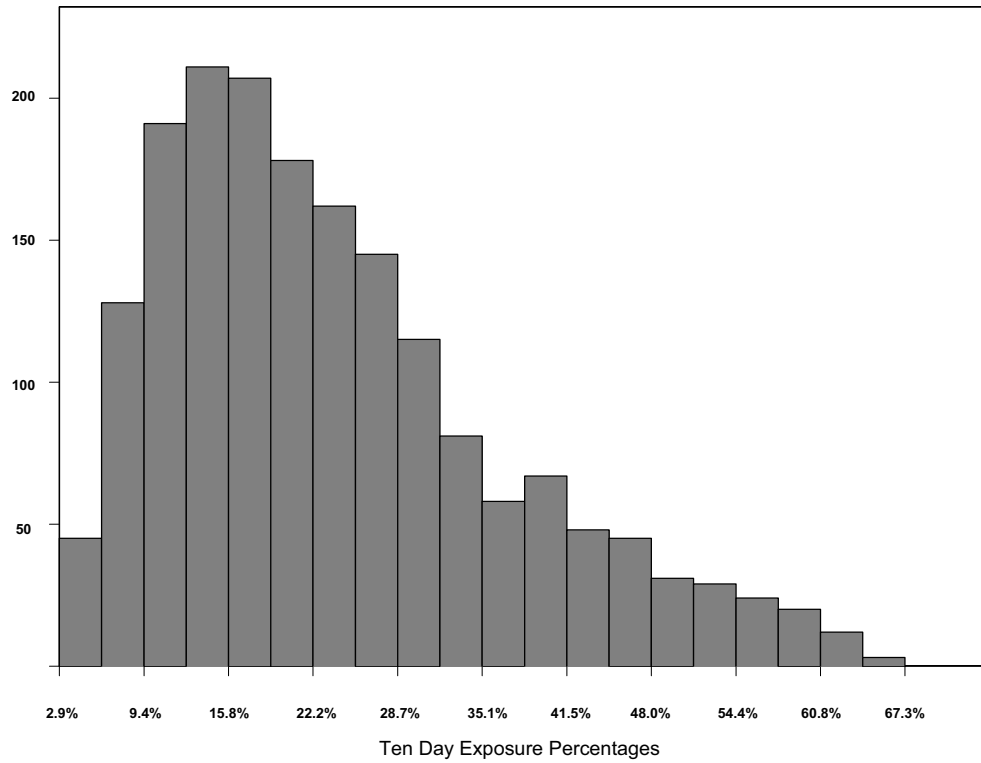


Figure 6. Histogram of Ten Day Exposure Percentages

2/10 there was actually a small drop). The rate of change is very steep in the beginning and then levels off as the incubation period increases. For the battalion command post as the incubation period went from 4 to 6 (from two times the latent period to three times the latent period), there was a 104% increase in total exposures. For the same unit, as the incubation period went from 6 to 8, there was only a 10% increase in the total number of exposures. The results were similar for an initial exposure of Alpha company at 25%. As the incubation period went from 4 to 6, there was a 110% increase in total exposures; from 6 to 8, there was a 13% increase in total exposures.

The goal of any weapon is to create the largest amount of casualties in the shortest amount of time. To that end a biological weapons designer would want a disease to spread quickly, but also become incapacitating in the shortest amount of time. In this data we have seen that by moving from an incubation period of 4 days

Latent and Incubation Period	BNCP @ 50% 10 Day Exp %	ACO @ 25% 10 Day Exp%
2/4	24.05%	14.21%
2/5	42.32%	24.27%
2/6	49.35%	29.91%
2/7	51.91%	30.91%
2/8	54.65%	33.93%
2/9	55.98%	34.11%
2/10	52.67%	36.56%

Table 2. Ten Day Exposure Percentages for Variations in Incubation Periods with Fixed Latent Period (3), Incubation Period(9) and Fixed Virulence (.67)

to one of 6 days we were able to double the effect of the weapon where as going from 4 days to 8 days gave only little additional effect.

## 2. Variations in Virulence

For the next scenario latent/incubation periods were fixed at 2/8, the unit and initial exposure level were fixed as the rear command post exposed at 25%. The total ten day exposure percentage was then examined for virulence .25, .33, .50, .67 and .75. The results can be found in Table 3.

Increasing virulence had interesting results. The first increases in virulence did not produce the largest increases in total exposures. Rather the middle increase from .33 to .50 produce the largest increase, an 81% increase in total exposures. The increases progressed as follows: Virulence .25 to .33, 44% increase in exposures; virulence .33 to .50, 81% increase in exposures; virulence .50 to .67, 41% increase in exposures; virulence .67 to .75, 8% increase in exposures. It appears as if the overall size of the epidemic is proportional to  $v(1 - v)$ .

One developing weapons then might strive for the .50 virulence and not put the effort into higher virulence at the expense of other aspects of the weapon. The time, effort and money need to increase virulence might be channeled into more effective delivery systems to increase the dissemination of a weaker agent over a larger percent

of the target population.

<b>Virulence</b>	<b>Ten Day Exposure %</b>
.25	12.45%
.33	17.93%
.50	32.57%
.67	46.02%
.75	49.85%

Table 3. Ten Day Exposure Percentages for Variations in Virulence with Fixed Latent Period (3), Incubation Period(9) and fixed Unit (BNRR @25%)

### 3. Variations in Units and Exposure Levels

The impact of which unit is initially infected and at what level that unit is infected goes far beyond the total number of exposures. Exposing one of the rifle companies will most likely take away one fourth of the combat units available to the commander. Exposing the battalion command post will disrupt command and control and take away many of the unit’s leaders. As medical, maintenance, communication, ammunition and armory support all lie inside the rear command post, an exposure here will disrupt nearly all service and support.

The results found in Table 4 reveal consistent changes in the overall number of exposed, as unit and exposure levels change. Doubling the initial level of exposure resulted in a mean growth of 51% in total exposures. In essence, doubling the dissemination capabilities increased the overall number of exposed  $1\frac{1}{2}$  times. There were no significant surprises in the differences of exposure based on which unit was initially exposed. The rear command post, which contains the bulk of the service and support units, facilitated the fastest spread of the epidemic. Weapons company which is the smallest unit and often operates independently of the other rifle companies facilitated the slowest spread of disease.

Unit	Level of Exposure	Ten Day % Exposure %
BNCP	25%	14.32%
BNCP	50%	23.57%
BNRR	25%	19.22%
BNRR	50%	29.16%
ACO	25%	14.32%
ACO	50%	21.69%
CCO+	25%	20.34%
CCO+	50%	27.49%
WepCO	25%	12.16%
WepCO	50%	18.91%

Table 4. Ten Day Exposure Percentages for Variations in Units and Exposure Levels, with Fixed Latent Period (3), Incubation Period (9) and Fixed Virulence (.67)

## B. COUNTERMEASURES

The previous section allowed the epidemic to spread unhindered for ten days. In this section four defensive techniques or countermeasures will be implemented to see if any impact can be made on the overall size of the epidemic. Countermeasures will include quarantine, elevation of the protective posture of troops, a limited quarantine strategy and a combination of two of these. The "Variations in Virulence" section will be the scenario used in testing the countermeasures. Latent and incubation periods have been fixed at 2 and 8 respectively and the rear command post will be exposed at 25%.

### 1. Countermeasure 1

A unit wide quarantine will be imposed on any unit that has members displaying active, diagnosable symptoms of disease. This will be done by setting all entries for this unit in the interaction matrix to zero and reducing the effective contact rate within this unit to zero. This knee-jerk reaction may effect the spread of the disease, but it will also have drastic effects on the ability of the BLT to conduct its mission. Completely quarantining the battalion command post will allow for command and

control only across the radio. Completely quarantining the service and support units will cause all resupply, meal and medical service to cease. This countermeasure will only be able to be done on a limited scope and for a short period of time. For this model a quarantine was imposed on a unit as soon as the first member of the unit reached the symptomatic stage ( $R(t)$ ).

For this scenario the first symptomatic individuals appeared on day 6 or 7. Applying the quarantine as units presented as symptomatic effectively cut the total number of individuals exposed to the disease by more than 32% (See Table 5). As these tests ran for just ten days the quarantine was short in duration but the problem was most definitely not solved. The quarantine strategy may be a good first measure as the BLT tries to identify the extent of the spread of the disease. This posture would not be viable for an extended period of time.

Another problem with this is that on average 19 platoons and 9 percent of the total population have at least been exposed to the disease by the time the first exposures become symptomatic. While one unit was being quarantined 18 others were still capable of spreading the disease throughout the BLT.

<b>Virulence</b>	<b>Unhindered Exposure %</b>	<b>Countermeasure 1 Exposure %</b>	<b>Reduction in Total Exposed</b>
.25	12.45%	7.18%	42.33%
.33	17.93%	12.00%	33.07%
.50	32.57%	21.21%	34.88%
.67	46.02%	31.32%	31.82%
.75	49.85%	39.44%	20.88%
<b>Average Reduction in Total Exposed</b>			<b>32.60%</b>

Table 5. Results from Countermeasure 1

## 2. Countermeasure 2

Mission oriented protective posture (MOPP) is the countermeasure with which combat troops are most familiar. From very early on in introductory training, troops



are taught that the first response to a nuclear, biological or chemical (NBC) attack is to don their protective gear. For this scenario the entire BLT will be placed at MOPP-4, the highest protective posture afforded to combat troops, when it becomes apparent there has been a biological attack ( $R(t) \geq 1$ ). This level of protection consists of wearing the entire protective suit to include rubber boots, gloves and the protective gas mask. The reality is that operating at MOPP-4 is difficult and taxing on troops. Basic functions such as drinking water, eating, using the latrine and using weapons becomes decidedly more difficult while at MOPP-4. The current threat from a biological weapon that has started an epidemic in the BLT might be able to be treated differently. The protective suit troops carry into combat protects against chemical agents and biological that have the ability to enter the body through unbroken skin. The biological agents that spread through person to person contact rely on spreading through respiratory droplets and other body fluids. The protective posture may be able to be relaxed and adapted to limit the disease's ability to spread while still allowing the troops to complete their mission (see Table 6).

<b>Virulence</b>	<b>Unhindered Exposure %</b>	<b>Countermeasure 2 Exposure %</b>	<b>Reduction in Total Exposed</b>
.25	12.45%	6.89%	44.66%
.33	17.93%	9.24%	48.47%
.50	32.57%	18.37%	43.60%
.67	46.02%	23.69%	48.52%
.75	49.85%	27.52%	44.79%
<b>Average Reduction in Total Exposed</b>			<b>46.01%</b>

Table 6. Results from Countermeasure 2

With an average reduction of over 46% , placing the unit into MOPP-4 proved to significantly reduce the spread of the disease. This reduction was reached without having to stop the interactions between units. Command and control can still take place face-to-face, resupply and rearming will be able to keep the units mission capable. The covert attack we are modeling here still allows for a significant spread of

the disease. Both of the previous countermeasures allow for action to be taken only after the symptoms of the disease become evident.

### 3. Countermeasure 3

If the intelligence community received information of an increased threat of attack by biological weapons, there may be time to implement a limited vaccination campaign. There are entire studies that investigate the most effective vaccination strategies. For the purpose of this study we simulate vaccinating a unit by reducing their effective contact rates by 85%. This would rely on knowing what the suspected agent would be, having the vaccines on hand and having time to let the vaccines become effective. We will assume for this case the all have been met and there are a limited number of vaccinations available. The decision has been made to vaccinate all of the service and support personnel as they are the most likely to spread the disease. Results for this countermeasure can be found in Table 7

<b>Virulence</b>	<b>Unhindered Exposure %</b>	<b>Countermeasure 3 Exposure %</b>	<b>Reduction in Total Exposed</b>
.25	12.45%	10.65%	14.46%
.33	17.93%	14.93%	16.73%
.50	32.57%	27.00%	17.10%
.67	46.02%	42.45%	7.76%
.75	49.85%	48.31%	3.01%
<b>Average Reduction in Total Exposed</b>			<b>11.81%</b>

Table 7. Results from Countermeasure 3

Although this strategy did reduce the total size of the epidemic by an average of more than 10%, the results were far less encouraging than the previous two countermeasures. The results are not completely unexpected though. Only 12% of the total population received the vaccination, this resulted in a decrease of nearly 12% in the size of the epidemic. Each of these countermeasures by itself has serious shortfalls

in either protection or the ability of the BLT to conduct its mission. A combination of countermeasures may provide a better solution.

#### 4. Countermeasure 4

This countermeasure combined a limited vaccination strategy with an increase in protective posture after the first symptoms appeared. This essentially combined countermeasures 2 and 3. As expected, combining two countermeasures increased the overall protection afforded to the BLT, consequently the epidemic was contained even further.

Realistically our forces need to have numerous options for protection against biological weapons. Both prophylactic and active protection can combine to give better protection than any one measure by itself. For this combination, the spread of the epidemic was cut, on average, by more than half. The 51.72% (see Table 8) decrease in overall size of the epidemic equates to 473 (34% of the population) fewer exposures in the worst case seen in this study.

Virulence	Unhindered Exposure %	Countermeasure 4 Exposure %	Reduction in Total Exposed
.25	12.45%	6.24%	49.88%
.33	17.93%	7.99%	55.44%
.50	32.57%	14.14%	56.59%
.67	46.02%	23.61%	48.70%
.75	49.85%	25.93%	47.98%
Average Reduction in Total Exposed			51.72%

Table 8. Results from Countermeasure 4

### C. ANALYSIS OF DIFFERENCE BETWEEN COUNTERMEASURES AND UNHINDERED SPREAD

To ensure that the difference found in applying the different countermeasures was not a simple statistical anomaly, Student's t-tests for paired means were applied to the data. With a null hypothesis that there was no difference between the two,

the one-tailed tests yielded probabilities much less than 1%. This essentially ensures that the countermeasures are having an effect on the size of the epidemic. The Excel produced analysis for countermeasures 1-3 can be seen in Fig. 7

T-Test analysis for Countermeasures versus Unhindered Spread					
L/I = 2/8	Vir .25				
Counter	Unhindered	t-Test: Paired Two Sample for Means			
Measure1	Spread				
8.66%	11.38%		Variable 1	Variable 2	
5.80%	12.11%	Mean	0.071806167	0.124375918	
8.22%	11.60%	Variance	0.000206841	0.000120914	
7.71%	13.07%	Observations	5	5	
5.51%	14.02%	Pearson Correlation	-0.68225238		
		Hypothesized Mean Difference	0		
		df	4		
		t Stat	-5.04200348		
		P(T<=t) one-tail	0.00363591		
		t Critical one-tail	2.131846486		
		P(T<=t) two-tail	0.00727182		
		t Critical two-tail	2.776450856		
L/I = 2/8	Vir 67				
Counter	Unhindered	t-Test: Paired Two Sample for Means			
Measure2	Spread				
24.23%	44.13%		Variable 1	Variable 2	
26.21%	50.44%	Mean	0.233333333	0.46020558	
21.00%	46.55%	Variance	0.000396055	0.000711412	
22.25%	44.05%	Observations	5	5	
22.98%	44.93%	Pearson Correlation	0.579836365		
		Hypothesized Mean Difference	0		
		df	4		
		t Stat	-22.8732062		
		P(T<=t) one-tail	1.08218E-05		
		t Critical one-tail	2.131846486		
		P(T<=t) two-tail	2.16436E-05		
		t Critical two-tail	2.776450856		
L/I = 2/8	Vir .50				
Counter	Unhindered	t-Test: Paired Two Sample for Means			
Measure3	Spread				
25.84%	30.76%		Variable 1	Variable 2	
26.80%	33.70%	Mean	0.270044053	0.325697504	
25.40%	33.55%	Variance	0.000351312	0.000441391	
30.18%	34.88%	Observations	5	5	
26.80%	29.96%	Pearson Correlation	0.516641297		
		Hypothesized Mean Difference	0		
		df	4		
		t Stat	-6.33561385		
		P(T<=t) one-tail	0.001588809		
		t Critical one-tail	2.131846486		
		P(T<=t) two-tail	0.003177617		
		t Critical two-tail	2.776450856		

Figure 7. t-test analysis of differences between countermeasures and unhindered spread

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## V. SUMMARY AND RECOMMENDATIONS

### A. SUMMARY

The threat of biological weapons is a very real threat to our military forces. While everyone is concerned about the possible consequences of a biological agent being released, the operating force in general is not prepared to deal with an outbreak of a contagious and deadly or disabling disease. The goal of this thesis was not only to develop a model to describe the spread of disease in a functioning military unit but also to develop a tool that might be useful to military planners at the tactical level.

If a military unit was covertly exposed to a biological weapon with the right combination of virulence, latent and incubation periods, initial target and exposure levels the unit can quickly become overwhelmed by the disease. In fifteen different trials over 60% (820 men) of the BLT became exposed to the disease after just 10 days. These numbers would completely overwhelm the medical services of the BLT and supporting Naval forces, the BLT would be essentially combat ineffective and the psychological impact on the Nation would be extreme.

Some basic countermeasures can be taken to reduce the spread of the disease. As examples quarantine, protective postures, incubation and combinations of these were tested to validate the model created. Each had a positive impact on the overall size of the epidemic.

The problem of modeling the impact of biological weapons is one that needs to be addressed by a much wider community. There are current studies and programs ongoing to do just this. The Joint Operational Effects Federation (JOEF) in its mission statement spells out the task at hand.

The requirement exists for a modeling and simulation (M&S) analytical capability to determine and assess the impact of nuclear, biological chemical and radiological warfare (NBCRW) on military operations. This requirement for an accredited, predictive, M&S capability supports both a near term re-

quirement for advance planning and analysis role supporting wartime operations and the far term requirement for near real-time decision making capabilities (i.e. combat).[Ref. 17]

## **B. RECOMMENDATIONS**

The task of developing a realistic model will be a daunting but necessary task. The issue will be getting the right combination of individuals working on the project. At a minimum representatives from the medical, operations analysis, tactical planning and mathematics communities need to come together to work on this project.

The model needs to be easy enough for tactical forces to apply in a field environment but also complete enough to produce quality information to the commander and his planning staff. The model developed in this research was Microsoft Excel based, all efforts should be made to keep the programs involved simple. Tactical forces do not need to learn an entirely new system for running these models.

This model defined a largest homogeneous unit (the platoon), there is no reason this model could not be expanded to be able to work with individual members of the unit. Experiments would need to be run to determine the exact interaction behavior down to the individual. The model could then combine that information along with the Table of Organization for the unit. The Table of Organization is a list of all of the individual billets within a unit. A program could retrieve both pieces of information and describe the spread of disease with much greater accuracy.

The modeling tool developed for this research was created in a relatively short period of time. Given ample time and the right combination of contributors, a simple yet effective planning tool could be developed to be used by tactical units. More time could then be devoted to developing the type of system the JOEF plans on fielding by 2008.

## APPENDIX. PROGRAMS FOR EPIDEMIC PROGRESSION

```
Sub InteractionMatrix()  
,  
' InteractionMatrix Macro  
' Macro recorded 1/22/2002 by rwpaters  
,  
  
' This Macro takes the initial population and infective agent  
' information and creates all other sheets needed to run the  
' model. The Interaction Matrix Sheet is a lower triangular  
' matrix where the user will enter the number of daily interactions  
' between two units. The Temp Sheet tracks and totals the number  
' of daily new exposures as a result of the cross-unit contamination.  
' This sheet will be hidden from view unless the user decides to  
' view it. The Epidemic Progression Sheet tracks the spread of the  
' disease. Daily numbers of susceptible, exposed, infectious and  
' removed from each unit along with population totals and day of  
' exposure is tracked.  
  
,  
  
'Creates Interaction Matrix Sheet  
  
    Sheets.Add  
    ActiveSheet.Name = "Interaction Matrix"  
    For i = 2 To 251  
        If Sheets("Population Characteristics").Cells(i, 2) <> "" Then  
            Sheets("Interaction Matrix").Cells(i, 1) = Sheets("Population_  
Characteristics").Cells(i, 2)  
            Sheets("Interaction Matrix").Cells(1, i) = Sheets("Population_  
Characteristics").Cells(i, 2)  
        End If  
    Next i  
    Rows("1:1").Select  
    With Selection  
        .HorizontalAlignment = xlGeneral
```



```

        .VerticalAlignment = xlBottom
        .WrapText = False
        .Orientation = 90
        .AddIndent = False
        .ShrinkToFit = False
        .MergeCells = False
    End With
    Selection.Columns.AutoFit

    For i = 2 To 251

        For J = i To 251
            If Cells(1, J) <> "" Then
                Cells(i, J).Select
                With Selection.Interior
                    .ColorIndex = 16
                    .Pattern = xlSolid
                    .PatternColorIndex = xlAutomatic
                End With
            End If
        Next J

        If Cells(i, 1) <> "" Then
            For J = 2 To i
                Cells(i, J) = Sheets("test").Cells(i, J)
            Next J
        End If

    Next i

'Creates Epidemic Progression Sheet

Sheets.Add
ActiveSheet.Name = "Epidemic Progression"
For i = 2 To 251
    If Sheets("Population Characteristics").Cells(i, 2) <> "" Then
        Sheets("Epidemic Progression").Cells(i, 1) = Sheets("Population_
Characteristics").Cells(i, 2)
    End If

```

```

    Sheets("Epidemic Progression").Cells(i, 2) = Sheets("Population_
Characteristics").Cells(i, 3)

    End If
Next i
Range("B1").Select
ActiveCell.FormulaR1C1 = "Size"
Range("C1").Select
ActiveCell.FormulaR1C1 = "S(t)"
Range("D1").Select
ActiveCell.FormulaR1C1 = "E(t)"
Range("E1").Select
ActiveCell.FormulaR1C1 = "I(t)"
Range("F1").Select
ActiveCell.FormulaR1C1 = "R(t)"
Range("G1").Select
ActiveCell.FormulaR1C1 = "% Available"
Columns("G:G").ColumnWidth = 12
Range("B1:G1").Select
Selection.Font.Bold = True
For J = 2 To 251
    If Cells(J, 2) <> "" Then
        Cells(J, 7).FormulaR1C1 = "=1 - RC[-1]/RC[-5]"
        Cells(J, 3).FormulaR1C1 = "=RC[-1]-RC[1]-RC[2]-RC[3]"
    End If
Next J
L = Sheets("Agent Characteristics").Cells(6, 2)
C = Sheets("Agent Characteristics").Cells(7, 2) - Sheets("Agent_
Characteristics").Cells(6, 2)

For i = 1 To L + 1
Cells(1, 14 + i) = "E" & i
Next i
For i = 1 To C + 1
Cells(1, 14 + L + 1 + i) = "I" & i
Next i

' Columns("O:IV").Select
' Selection.EntireColumn.Hidden = True

```

```

' Creates Temp Sheet

Sheets.Add
ActiveSheet.Name = "Temp"
For i = 2 To 251
    If Sheets("Population Characteristics").Cells(i, 2) <> "" Then
        Sheets("Temp").Cells(i, 1) = Sheets("Population_
Characteristics").Cells(i, 2)
        Sheets("Temp").Cells(1, i) = Sheets("Population_
Characteristics").Cells(i, 2)
    End If
Next i
Rows("1:1").Select
With Selection
    .HorizontalAlignment = xlGeneral
    .VerticalAlignment = xlBottom
    .WrapText = False
    .Orientation = 90
    .AddIndent = False
    .ShrinkToFit = False
    .MergeCells = False
End With
Selection.Columns.AutoFit
Worksheets("Epidemic Progression").Activate
For i = 2 To 7
    For J = 1 To 2
        Cells(i + 18, J + 8) = Sheets("Agent Characteristics")_
.Cells(i, J)
    Next J
Next i
Columns("I:I").Select
Selection.Columns.AutoFit
Range("I20:I26").Select
With Selection.Interior
    .ColorIndex = 15
    .Pattern = xlSolid
    .PatternColorIndex = xlAutomatic
End With
Cells(4, 10) = "Day"
Cells(4, 11) = "1"
Cells(5, 10) = "Population Totals"
Cells(4, 13) = "N ="

```

```
Cells(9, 11) = "#Units Exp"
For i = 10 To 13
Cells(6, i) = Cells(1, i - 7)
Next i
```

```
ActiveSheet.Buttons.Add(405.75, 45.75, 50, 72).Select
Selection.OnAction = "EpidemicProgress"
i = ActiveSheet.Shapes.Count
ActiveSheet.Shapes(i).Select
Selection.Characters.Text = "Advance Time"
With Selection.Characters(Start:=1, Length:=15).Font
    .Name = "Arial"
    .FontStyle = "Regular"
    .Size = 10
    .Strikethrough = False
    .Superscript = False
    .Subscript = False
    .OutlineFont = False
    .Shadow = False
    .Underline = xlUnderlineStyleNone
    .ColorIndex = xlAutomatic
End With
Selection.ShapeRange.ScaleWidth 1.58, msoFalse, msoScaleFromTopLeft
Selection.ShapeRange.ScaleHeight 0.26, msoFalse, msoScaleFromTopLeft
```

```
ActiveSheet.Buttons.Add(405.75, 95.75, 50, 72).Select
Selection.OnAction = "Reset"
i = ActiveSheet.Shapes.Count
ActiveSheet.Shapes(i).Select
Selection.Characters.Text = "Reset this page"
With Selection.Characters(Start:=1, Length:=15).Font
    .Name = "Arial"
    .FontStyle = "Regular"
    .Size = 10
    .Strikethrough = False
    .Superscript = False
    .Subscript = False
    .OutlineFont = False
    .Shadow = False
    .Underline = xlUnderlineStyleNone
    .ColorIndex = xlAutomatic
```

```

End With
Selection.ShapeRange.ScaleWidth 1.58, msoFalse, msoScaleFromTopLeft
Selection.ShapeRange.ScaleHeight 0.26, msoFalse, msoScaleFromTopLeft

ActiveSheet.Buttons.Add(405.75, 145.75, 50, 110).Select
Selection.OnAction = "Resetall"
i = ActiveSheet.Shapes.Count
ActiveSheet.Shapes(i).Select
Selection.Characters.Text = "Reset Entire Program"
With Selection.Characters(Start:=1, Length:=15).Font
    .Name = "Arial"
    .FontStyle = "Regular"
    .Size = 10
    .Strikethrough = False
    .Superscript = False
    .Subscript = False
    .OutlineFont = False
    .Shadow = False
    .Underline = xlUnderlineStyleNone
    .ColorIndex = xlAutomatic
End With
Selection.ShapeRange.ScaleWidth 1.58, msoFalse, msoScaleFromTopLeft
Selection.ShapeRange.ScaleHeight 0.26, msoFalse, msoScaleFromTopLeft

ActiveSheet.Buttons.Add(405.75, 195.75, 50, 72).Select
Selection.OnAction = "RecordData"
i = ActiveSheet.Shapes.Count
ActiveSheet.Shapes(i).Select
Selection.Characters.Text = "Record Action"
With Selection.Characters(Start:=1, Length:=15).Font
    .Name = "Arial"
    .FontStyle = "Regular"
    .Size = 10
    .Strikethrough = False
    .Superscript = False
    .Subscript = False
    .OutlineFont = False
    .Shadow = False
    .Underline = xlUnderlineStyleNone
    .ColorIndex = xlAutomatic
End With
Selection.ShapeRange.ScaleWidth 1.58, msoFalse, msoScaleFromTopLeft

```

```

        Selection.ShapeRange.ScaleHeight 0.26, msoFalse, msoScaleFromTopLeft

Worksheets("test").Visible = False
Worksheets("Temp").Visible = False

End Sub
' -----
' -----

Sub EpidemicProgress()
'
' EpidemicProgress Macro
' Macro recorded 1/22/2002 by rwpaters
' This macro advances the daily progression of the epidemic in three
' phases. First the previous day's data is shifted and new exposures
' from the homogeneous (intraunit) spread is computed. Next heterogeneuos
' (interunit) spread is computed and last they are combined and recorded
' as the current days entries.

Worksheets("Epidemic Progression").Activate
L = Sheets("Agent Characteristics").Cells(6, 2)
C = Sheets("Agent Characteristics").Cells(7, 2) - Sheets("Agent_
Characteristics").Cells(6, 2)

' -----
'Sets intial exposure or infection into action
If Cells(4, 11) = 1 Then
    For i = 2 To 251
        If Cells(i, 2) <> "" Then
            Cells(i, 16 + L) = Cells(i, 5)
            Cells(i, 15) = Cells(i, 4)
        End If

        If Cells(i, 4) <> "" Then
            b = i
            For J = 2 To 251
                If Sheets("Data runs").Cells(J, 2) <> "" Then
                    a = J
                End If
            End For
        End If
    End For
End If

```

```

Next J
End If

Next i

Sheets("Data runs").Cells(a + 1, 1) = Cells(b, 1)
Sheets("Data runs").Cells(a + 1, 2) = "Initial Exposure"
Sheets("Data runs").Cells(a + 1, 3) = Sheets("Agent Characteristics")_
.Cells(6, 2) 'latent
Sheets("Data runs").Cells(a + 1, 4) = Sheets("Agent Characteristics")_
.Cells(7, 2) 'incubation
Sheets("Data runs").Cells(a + 1, 5) = Sheets("Agent Characteristics")_
.Cells(4, 2) 'virulence
Sheets("Data runs").Cells(a + 1, 6) = Sheets("Epidemic Progression")_
.Cells(4, 11) 'day
Sheets("Data runs").Cells(a + 1, 7) = Cells(b, 4) 'S(t)
Sheets("Data runs").Cells(a + 1, 8) = "0" 'E(t)
Sheets("Data runs").Cells(a + 1, 9) = "0" 'I(t)
Sheets("Data runs").Cells(a + 1, 10) = "0" 'R(t)
Sheets("Data runs").Cells(a + 1, 11) = "1" '#Units Exposed
Sheets("Data runs").Cells(a + 1, 12) = "0" '#Units Infected
Sheets("Data runs").Cells(a + 1, 13) = Cells(b, 4) / Cells(5, 13)

```

```
End If
```

```
' -----
```

```
For J = 2 To 251
```

```

If Cells(J, 2) <> "" Then
    t1 = Cells(J, 15) '
    Remd = Cells(J, 16 + L + C) '
    For i = 15 To 15 + L + C 'This area shifts day(t) information
        t2 = Cells(J, i + 1) 'to prepare for next set of
        Cells(J, i + 1) = t1 'calculations
        t1 = t2 '
    Next i '
    Cells(J, 17 + L + C) = Null '

```

```

' -----
'   Intraunit Spread Computation

'   New Exposures ~ S(t)*I(t)

    ecr = Norm(Sheets("Population Characteristics")_
.Cells(J, 4), 0.1)
    If ecr > 0 Then
        ExpN = Round((Cells(J, 3) * Cells(J, 5) * ecr * _
Norm(Cells(22, 10), 0.1) / Cells(J, 2)))
    Else
        ExpN = 0
    End If

'Once an individual enters the exposed stage the transition through the
' exposed, infectious and removed stage is an absorbing Markov process.
' Selects which individuals transition and when that transition occurs.
' Basic transition probabilities are that E_1 >> E_2 wp 1;
'E_i >> E_i+1 wp 1; E_L-2 >> E_L-1 wp 1; E_L-1 >> E_L wp .8,
'E_L-1 >> I_1 wp .2; E_L >> E_L+1 wp .4, E_L >> I_1 wp .6;
'E_L+1 >> I_1 wp 1; I_1 >> I_2 wp 1; I_i >> I_i+1 wp 1;
'I_L-2 >> I_L-1 wp 1; I_L-1 >> I_L wp .8, I_L-1 >> R wp .2;
'I_L >> I_L+1 wp .4, I_L >> R wp .6; I_L+1 >> R wp 1;

'New Infections (those transitioning to I_1)
e1 = 0
For i = 1 To Cells(J, 15 + L)
    a = Rnd()
    If a <= 0.6 Then
        e1 = e1 + 1
    End If
Next i

e2 = 0
For i = 1 To Cells(J, 14 + L)
    a = Rnd()
    If a <= 0.2 Then
        e2 = e2 + 1
    End If
Next i

```



```
Cells(J, 16 + L) = Cells(J, 16 + L) + e1 + e2
```

```
'Exposure adjustments
```

```
Cells(J, 15 + L) = Cells(J, 15 + L) - e1
```

```
Cells(J, 14 + L) = Cells(J, 14 + L) - e2
```

```
'New Removals (those transitioning to R)
```

```
r1 = 0
```

```
For i = 1 To Cells(J, 15 + L + C)
```

```
a = Rnd()
```

```
If a <= 0.6 Then
```

```
r1 = r1 + 1
```

```
End If
```

```
Next i
```

```
r2 = 0
```

```
For i = 1 To Cells(J, 14 + L + C)
```

```
a = Rnd()
```

```
If a <= 0.2 Then
```

```
r2 = r2 + 1
```

```
End If
```

```
Next i
```

```
RemT = Remd + r1 + r2
```

```
'Infections adjustment
```

```
Cells(J, 15 + L + C) = Cells(J, 15 + L + C) - r1
```

```
Cells(J, 14 + L + C) = Cells(J, 14 + L + C) - r2
```

```
'-----  
' The Heterogenous or interunit spread of the disease uses the user supplied  
' interaction matrix to determine how many effective contacts become new  
' exposures. This area randomly selects pairs of individuals from the two  
' concerned units and with probatibility = virulence, when an  
' infectious individual contacts a susceptible, a new exposure is created.
```

```
' Interunit Spread Computation
```

```

For i = J + 1 To 251
    If Sheets("Interaction Matrix").Cells(i, J) <> "" Then
        te = 0
        be = 0

        For h = 1 To Sheets("Interaction Matrix").Cells(i, J)
            t = 1 + Int(Cells(J, 2) * Rnd)
            b = 1 + Int(Cells(i, 2) * Rnd)
            If t <= Cells(J, 5) And b > Cells(i, 5) Then
                be = be + 1
            End If
            If b <= Cells(i, 5) And t > Cells(J, 5) Then
                te = te + 1
            End If
        Next h

        Sheets("Temp").Cells(i, J) = te
        Sheets("Temp").Cells(J, i) = be
    End If
Next i

```

```

ExpH = 0

```

```

For k = 2 To 251
    ExpH = ExpH + Sheets("Temp").Cells(k, J)
Next k

```

```

ExpH = Round(ExpH * Norm(Cells(22, 10), 0.1))

```

```

'-----
'Now the Homogeneous and Heterogeneous spread figures are combined
'to give the new end of day totals. These are then used for the next
'days computations.

```

```

' New Exposures Computation
If Cells(J, 3) >= ExpH + ExpN Then
    Cells(J, 15) = ExpH + ExpN
End If

If Cells(J, 3) < ExpH + ExpN Then

```

```

        Cells(J, 15) = Cells(J, 3)
    End If

    ExpT = 0
    For i = 15 To 15 + L
        ExpT = ExpT + Cells(J, i)
    Next i
    Cells(J, 4) = (ExpT)

    Inft = 0
    For i = 16 + L To 16 + L + C
        Inft = Inft + Cells(J, i)
    Next i
    Cells(J, 5) = (Inft)
    Cells(J, 6) = Cells(J, 6) + (RemT)

End If

Next J
' -----

' Entire Unit Totals
    ST = 0
    ET = 0
    IT = 0
    RT = 0
    UE = 0
    UI = 0
    For i = 2 To 251
        If Cells(i, 2) <> "" Then
            ST = ST + Cells(i, 3)
            ET = ET + Cells(i, 4)
            IT = IT + Cells(i, 5)
            RT = RT + Cells(i, 6)
            If Cells(i, 4) <> "0" Then
                UE = UE + 1
            End If
            If Cells(i, 5) <> "0" Then
                UI = UI + 1
            End If
        End If
    Next i

```

```

End If

Next i

Cells(7, 10) = ST
Cells(7, 11) = ET
Cells(7, 12) = IT
Cells(7, 13) = RT
Cells(5, 13) = ST + ET + IT + RT
Cells(10, 11) = UE
Cells(10, 13) = UI
Cells(4, 11) = Cells(4, 11) + 1

For i = 2 To 250
If Sheets("Data runs").Cells(i, 2) <> "" Then
a = i
End If
Next i
Sheets("Data runs").Cells(a + 1, 2) = "None"
Sheets("Data runs").Cells(a + 1, 3) = Sheets("Agent Characteristics")_
.Cells(6, 2) 'latent
Sheets("Data runs").Cells(a + 1, 4) = Sheets("Agent Characteristics")_
.Cells(7, 2) 'incubation
Sheets("Data runs").Cells(a + 1, 5) = Sheets("Agent Characteristics")_
.Cells(4, 2) 'virulence
Sheets("Data runs").Cells(a + 1, 6) = Sheets("Epidemic Progression")_
.Cells(4, 11) 'day
Sheets("Data runs").Cells(a + 1, 7) = Sheets("Epidemic Progression")_
.Cells(7, 10) 'S(t)
Sheets("Data runs").Cells(a + 1, 8) = Sheets("Epidemic Progression")_
.Cells(7, 11) 'E(t)
Sheets("Data runs").Cells(a + 1, 9) = Sheets("Epidemic Progression")_
.Cells(7, 12) 'E(t)
Sheets("Data runs").Cells(a + 1, 10) = Sheets("Epidemic Progression")_
.Cells(7, 13) 'R(t)
Sheets("Data runs").Cells(a + 1, 11) = Sheets("Epidemic Progression")_
.Cells(10, 11) '#Units Exposed
Sheets("Data runs").Cells(a + 1, 12) = Sheets("Epidemic Progression")_
.Cells(10, 13) '#Units Exposed
Sheets("Data runs").Cells(a + 1, 13) = (Sheets("Epidemic Progression")_
.Cells(7, 11) + Sheets("Epidemic Progression").Cells(7, 12))_

```

```

/ Sheets("Epidemic Progression").Cells(5, 13)

End Sub

'**------
Function Norm(mean As Double, sd As Double) As Double

'Returns one normal random variable from N(mean,sd) distn
'Utilizes polar method as described in Simulation Modeling
'and Analysis (Law & Kelton) page 491.

Dim w As Double, u1 As Double, u2 As Double, v1 As Double
Dim v2 As Double, x1 As Double, x2 As Double

w = 2
While w > 1
    u1 = Rnd()
    u2 = Rnd()
    v1 = 2 * u1 - 1
    v2 = 2 * u2 - 1
    w = v1 * v1 + v2 * v2
Wend

x1 = sd * v1 * Sqr(-2 * Log(w) / w) + mean
x2 = sd * v2 * Sqr(-2 * Log(w) / w) + mean

If (Rnd() < 0.5) Then    'Randomly choose one of two r.v.'s
    Norm = x1
Else
    Norm = x2
End If

End Function

Sub Resetall()
,
' Resetall Macro
' Macro recorded 2/7/2002 by rwpaters
' Resets the entire program back to the initial worksheet.
,

```

```

Sheets("Temp").Delete

Sheets("Epidemic Progression").Delete
Sheets("Interaction Matrix").Activate
For i = 2 To 251
If Cells(i, 1) <> "" Then
For J = 2 To i
Sheets("test").Cells(i, J) = Cells(i, J)
Next J
End If
Next i

Sheets("Interaction Matrix").Delete

Sheets("Data runs").Activate
For i = 2 To 250
If Cells(i, 2) <> "" Then
a = i
End If
Next i
Cells(a + 1, 2) = "RESET ALL"
For J = 1 To 13

Cells(a + 1, J).Select
With Selection.Interior
.ColorIndex = 56
.Pattern = xlSolid
.PatternColorIndex = xlAutomatic
End With

Next J
Sheets("Population Characteristics").Activate
End Sub

Sub Reset()
,
' Reset Macro
' Macro recorded 1/10/2002 by rwpaters
' Resets the Epidemic Progress sheet back to day = 0
,

L = Sheets("Agent Characteristics").Cells(6, 2)

```

```

    C = Sheets("Agent Characteristics").Cells(7, 2) - Sheets("Agent_
Characteristics").Cells(6, 2)
    For i = 2 To 251
        If Cells(i, 2) <> "" Then
            For J = 4 To 6
                Cells(i, J) = Null
            Next J
            For J = 15 To 16 + L + C
                Cells(i, J) = Null
            Next J
        End If
    Next i
    For i = 10 To 13
        Cells(7, i) = Null
    Next i
    Cells(4, 11) = "1"
    Cells(10, 11) = Null

    For i = 2 To 250
        Sheets("Data runs").Activate
        If Cells(i, 2) <> "" Then
            a = i
        End If
    Next i
    Cells(a + 1, 2) = "RESET"
    For J = 1 To 13

        Cells(a + 1, J).Select
        With Selection.Interior
            .ColorIndex = 15
            .Pattern = xlSolid
            .PatternColorIndex = xlAutomatic
        End With

    Next J
    Sheets("Epidemic Progression").Activate

End Sub

Sub RecordData()
    ,

```

```
' RecordData Macro
' Macro recorded 2/18/2002 by rwpaters
'
' Keyboard Shortcut: Ctrl+r
'
    Sheets("Data runs").Activate
    For i = 2 To 250
        If Sheets("Data runs").Cells(i, 2) <> "" Then
            a = i
        End If
    Next i
    Cells(a, 2).Select

End Sub
```



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# LIST OF REFERENCES

- [1] Denis Mollison, editor. *Epidemic Models: Their Structure and Relation to Data*. Cambridge University Press, Cambridge, 1995.
- [2] World Health Organization Group of Consultants. *Health Aspects of Biological and Chemical Weapons(unofficial draft)*. World Health Organization, Geneva, 2001.
- [3] Ken Alibek. *Biohazard*. Academic Press/Harcourt Brace Jovanovich, Boston, 1998.
- [4] Charles Hennekens and Julie E. Burning. *Epidemiology in Medicine*. Little, Brown and Company, Boston, 1987.
- [5] D.S. Jones and B.D. Sleeman. *Differential Equations and Mathematical Biology*. George, Allen and Unwin, Boston, 1983.
- [6] David W. Siegrist. Advanced technology to counter biological terrorism. A presentation to the International Conference on Threats in the Technological Age, Holn, Isreal, 1998.
- [7] Mark G. Kortepeter and Gerald W. Parker. Potential biological weapons threats. *Emerging Infectious Diseases*, 5(4):1–7, 1999.
- [8] Office of Technology Assessment U.S. Congress. *Proliferation of Weapons of Mass Destruction: Assessing the Risk*. OTA-ISC-559, U.S. Government Printing Office, Washington, DC, 1993.
- [9] Dr. Ken Alibek. Biological weapons threats and defense. A lecture presented at the Naval Postgraduate School, February 7, 2001.
- [10] Organization of marine corps forces. Marine Corps Reference Publication (MCRP) 5-12D,, 1998.
- [11] Herbet Hethcote. The mathematics of infectious diseases. *SIAM Review*, 42:599–653, 2000.
- [12] David G. Kendall. Deterministic and stochastic epidemics in closed populaitons. *Proc. Symp. Math. Statist. Probability, 3rd, Berkley, CA*, 4:149–165, 1956.
- [13] Norman T. J. Bailey. The total size of a general stochastic epidemic. *Biometrika*, 40:177–185, 1954.
- [14] J.P. Gabriel, C. Lefevre, and P. Picard. *Lecture Notes in Biomathematics*. Springer-Verlag, New York, 1988.

- [15] John Fox Lila Elveback and Eugene Ackerman. Stochastic simulation models for two immunization problems. *Epidemiology: Proceedings of a SIMS Conference on Epidemiology*, July 8-12:90–103, 1974.
- [16] John Bombardt Jr. Contagious disease dynamics for biological warfare and bioterrorism casualty assessments. *Institute for Defense Analyses; IDA Paper P-3488*, 2000.
- [17] Operational requirements document (ord) for joint operational effects federation (joef). Draft Publication, 2001.

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